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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.
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08/838,486 04/07/97 BAEKKESKOV

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TOWNSEND AND TOWNSEND AND CREW
TWO EMBARCADERO CENTER 8TH FLOOR
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EXAMINER

TUNG, M	
ART UNIT	PAPER NUMBER

1644

DATE MAILED:

04/24/01

21

Please find below and/or attached an Office communication concerning this application or proceeding.

Commissioner of Patents and Trademarks

Office Action Summary

Application No.
08/838,486

Applicant(s)
Baekkeskov, et al.

Examiner
Mary B. Tung

Art Unit
1644



-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136 (a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).
- Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

- 1) ☒ Responsive to communication(s) filed on Jan. 10, 2001
- 2a) ☒ This action is FINAL. 2b) ☐ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 35 C.D. 11; 453 O.G. 213.

Disposition of Claims

- 4) ☒ Claim(s) 31, 34, 35, 38-42, and 49-63 is/are pending in the application.
- 4a) Of the above, claim(s) 38-42 is/are withdrawn from consideration.
- 5) ☐ Claim(s) _____ is/are allowed.
- 6) ☒ Claim(s) 31, 34, 35, and 49-63 is/are rejected.
- 7) ☐ Claim(s) _____ is/are objected to.
- 8) ☐ Claims _____ are subject to restriction and/or election requirements.

Application Papers

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☐ The drawing(s) filed on _____ is/are objected to by the Examiner.
- 11) ☐ The proposed drawing correction filed on _____ is: a) ☐ approved b) ☐ disapproved.
- 12) ☐ The oath or declaration is objected to by the Examiner.

Priority under 35 U.S.C. § 119

- 13) ☐ Acknowledgement is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d).
- a) ☐ All b) ☐ Some* c) ☐ None of:
- ☐ Certified copies of the priority documents have been received.
 - ☐ Certified copies of the priority documents have been received in Application No. _____.
 - ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).
- *See the attached detailed Office action for a list of the certified copies not received.
- 14) ☐ Acknowledgement is made of a claim for domestic priority under 35 U.S.C. § 119(e).

Attachment(s)

- 15) ☒ Notice of References Cited (PTO-892)
- 16) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
- 17) ☐ Information Disclosure Statement(s) (PTO-1449) Paper No(s) _____
- 18) ☐ Interview Summary (PTO-413) Paper No(s) _____
- 19) ☐ Notice of Informal Patent Application (PTO-152)
- 20) ☐ Other: _____

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DETAILED ACTION

1. Claims 1-30 and 43-48 were cancelled in the preliminary amendment filed April 7, 1997.
2. Claims 32, 33, 36 and 37 have been cancelled in the amendment filed Nov. 2, 1998 (Paper No. 9).
3. Non-elected claims 38-42 were withdrawn from consideration by the Examiner in the paper mailed April 28, 1998 (Paper No. 6).
4. Claims 49-57 were added in the amendment filed Nov. 2, 1998, Paper No. 9.
5. Claims 58 and 59 were added in the amendment filed 7/28/99, Paper No. 13.
6. Claims 60 and 61 were added in the paper filed 4/14/00, Paper No. 17.
7. Claims 31, 34, 35, 38-42, 49-61 are pending in this application.
8. Claims 62 and 63 were added in the paper filed 1/10/2001, Paper No. 20.
9. The Examiner acknowledges the comments by Mr. Liebeschuetz, concerning the comments in Paper No. 20 (Page 2, numbered paragraph 8), in reference to the interview with Applicants' representative and Biotech Specialist Richard Schwartz, SPE Christina Chan and Examiner Mary Tung on 3/21/00. The Examiner maintains her previous comments.
10. The Applicants maintain that the claims in the instant application are patentably indistinguishable from "the Florida patents". As all issued patents are presumed valid, the Examiner maintains that she will not comment in this or in any other action about the validity or other matters concerning the issued patents, nor on pending applications, as required under 35 U.S.C. 122 and 37 C.F.R. 1.14. Upon this basis, the Applicants question the presence of the signature of the Group Director on the previous action. As the Applicants indicated that claims 60 and 61 were copied, in accordance with 37 C.F.R. 1.607(c) (*MPEP 2001.06(d)*), from an issued patent (US Patent No. 5,762,937), it was determined that a Group Director's signature was desirable.
11. The Applicants argue in paragraph 11 of page 3 of Paper No. 20, that the "Examiner has not said why she believes there is a difference in scope" between the instant and the '937 patent. "If the Examiner is correct that the present claims are patentably distinct from the claims in the '937 patent (and the '360 patent), then Applicants can overcome these patent[s] by supplying evidence of derivation by declaration rather than interference." [brackets added by Examiner] If it is the understanding of the Examiner that Applicants are asserting on the

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record that all the instant claims are patentably indistinct from copied claims 60 and 61, then the pending claims would be subject to rejection under 35 U.S.C. 103(a) as being obvious over copied claims 60 and 61. Additionally, the issue of the difference in scope was addressed over the '937 (and 6,001,360) patent in the new matter rejection, as claims 60 and 61 were copied from the '937 patent. The 08/455,725 (properly 08/485,725) Tobin, et al. application discussed by Applicants in Paper No. 17 has issued as US Patent No. 5,998,366, which is cited as art not relied upon below.

Claim Rejections - 35 U.S.C. § 112

12. The following is a quotation of the first paragraph of 35 U.S.C. 112:

The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

13. Applicant's arguments filed in Paper No. 20 have been fully considered but they are not persuasive.

14. Claims 31, 34, 35, 49-59, 60 and 61 and newly-added claims 62 and 63 stand rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention.

15. The goal of peptide immunotherapy of T-cell-mediated autoimmunity is to induce anergy in self reactive T cells. Therefore, the pathologies of autoreactive T cells in autoimmunity can be blocked by using the appropriate autoantigen or autoantigen-derived peptides (see Tisch, et al., (*Proc. Natl. Acad. Sci. USA* 91:437-438, 1994), page 437, col. 1, in particular). However, the effectiveness of this therapy hinges on several factors: one is whether the therapy can be used to treat an ongoing autoimmune response or whether it is useful only in preventing the disease. Typically, an autoimmune disease is diagnosed at the time of onset when significant tissue damage has already occurred. The onset of IDDM is not predictable and therefore, prophylaxis of these diseases is not currently possible; currently, therapy is initiated in these conditions only after the onset of disease symptoms. Furthermore, Tisch et al., (*Proc. Natl. Acad. Sci. USA* 91:437-438, 1994) teach that treating an ongoing T-cell-mediated autoimmunity by administering an antigen peptide may have an immunizing effect and exacerbate the disease condition ((*Proc. Natl. Acad. Sci. USA* 91:437-438, 1994), page 437, column 3, in particular). How the antigen is administered is also a key factor in determining whether an immunogenic or tolerogenic response is induced. The duration of the toleragenic effect is an additional factor. Frequent treatment over a prolonged

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period of time may result in unforeseen immunological complications. Furthermore, the Applicant discloses on page 20, lines 16-19, that "care should be taken that administration of the pharmaceutical compositions of the present invention does not potentiate the autoimmune response." There is a lack of guidance in the specification as to how the potentiation of the autoimmune response would be prevented using the instant invention. Additionally, the high degree of specificity required for the process of clonal deletion/anergy may be limiting when dealing with diseases such as MS, IDDM and rheumatoid arthritis, in which there are responses to several antigens ((Tisch, et al., *Proc. Natl. Acad. Sci. USA* 91:437-438, 1994), see page 437, col. 2 ¶ 3 and bridging over to col. 3, ¶ 4). Additionally, Lernmark (*J. Int. Med.* 240:259-277, 1996) teaches that "The mechanisms of GAD65-induced protection of spontaneous diabetes is critical to our understanding of autoimmune diabetes. Further experiments also extended to the spontaneously diabetic BB rat are warranted to determine the mechanism of protection, especially as other investigators have not found the published procedures to be easily reproducible." ((*J. Int. Med.* 240:259-277, 1996), see page 274, col. 2, paragraph 1, in particular). Additionally, Harrison (*Molec. Med.* 1:722-727, 1995) teaches that "Insulin and GAD are strong candidate tolerogens for the prevention of human IDDM. However, caution should be exercised with GAD because, unlike insulin, it is not β cell specific and is found in high concentrations in the brain as well as in peripheral tissues other than islets. Without further animal studies and knowledge of the GAD epitopes that elicit T cell reactivity unique to human β cells, it would seem unwise to manipulate immunity to this widely distributed key enzyme. For the present, insulin (or proinsulin) is the only islet antigen that, both on scientific and ethical grounds, justifies therapeutic application to humans at risk of IDDM." ((*J. Int. Med.* 240:259-277, 1996), see page 724, col. 2, paragraph 2, in particular). Applicant has provided only in vitro experiments demonstrating the identity of GAD in rat islets of Langerhans and in rat brain, and anti-GAD antibodies in the sera of patient with IDDM and stiff man syndrome, to demonstrate operability of the claimed polypeptide. Since human and rats display different major histocompatibility complex haplotypes and Applicant has given no guidance as to how their peptide specific therapy would overcome autoreactive T cell escape mechanisms in humans or whether the peptide would induce autoimmunity or tolerance, it would require an undue amount of experimentation to one of skill in the art to practice the claimed invention.

16. The Applicants provided a Diamyd Corporation Press Release (12/21/2000; www.diamyd.com/docs/000118en.html) that teaches an FDA Phase I trial of the use of GAD65. The trial outcome was that there were no adverse clinical effect and auto antibodies to GAD65 were not detected during the study period. However, the study does not address the concerns raised by the Examiner based upon the teachings by Tisch, Lernmark and Harrison that GAD administration could cause adverse effects long term and how the treatment would be used to prevent diabetes. The portion of the

study identifying a "successful Diamyd vaccine" is speculative and admittedly "forward looking", and thus is not persuasive.

17. The Applicants further argue that the "Examiner appears to overlook the fact that the application discloses that onset of IDDM can be predicted from the presence of autoantibodies to GAD in the serum." This argument does not address whether the therapy can work, after autoantibodies have already formed. Also, if the Applicants assertions that the autoantibodies to GAD are the cause of IDDM, then the Examiner maintains that the disease has already started when such antibodies are detected, even though clinical symptoms may not be manifested. Therefore, the Applicants have failed to provide sufficient evidence that they can prevent the disease or treat it, once the autoantibodies have formed.
18. The Applicants further argue that the Examiner is criticizing the application for not disclosing dosages or routes of administration. The Examiner finds this argument confusing, as she is unable to locate such a requirement or criticism in previous actions. However, in the original rejection (reproduced, supra), the Examiner stated: "Furthermore, Tisch et al., (*Proc. Natl. Acad. Sci. USA* 91:437-438, 1994) teach that treating an ongoing T-cell-mediated autoimmunity by administering an antigen peptide may have an immunizing effect and exacerbate the disease condition ((*Proc. Natl. Acad. Sci. USA* 91:437-438, 1994), page 437, column 3, in particular). How the antigen is administered is also a key factor in determining whether an immunogenic or tolerogenic response is induced. The duration of the toleragenic effect is an additional factor. Frequent treatment over a prolonged period of time may result in unforeseen immunological complications. Furthermore, the Applicant discloses on page 20, lines 16-19, that "care should be taken that administration of the pharmaceutical compositions of the present invention does not potentiate the autoimmune response." There is a lack of guidance in the specification as to how the potentiation of the autoimmune response would be prevented using the instant invention." Thus, according to Tisch and the Applicant's own specification, how the antigen is administered is a "key factor". The Applicants have not addressed this point, either in the specification or in their subsequent remarks.
19. The applicants further argue that the Examiner does not address the Applicants' response to the existence of multiple autoantigens in diseases such as IDDM. The Applicants maintain that since a T cell response to GAD65 develops early in the development of IDDM and "subsequently spreads to other β cell antigens in a cascade of responses that ultimately lead to IDDM" that it "would be expected that inducing tolerance to GAD65 would abort subsequent events in the cascade of events leading to IDDM." The Applicants further point to several studies in which they assert the administration of GAD65 inhibits the development of IDDM in laboratory animals. These studies are discussed below. In short, the analysis of the various animal models for use in IDDM taught in literature cited by the Examiner discuss that each model has limitations and the models and interpretations are not limited to NOD mice.
20. The Applicants also argue that the Lernmark reference is being given undue emphasis in light of the numerous other reference provided by the Applicants in support of their

invention. However, the Examiner maintains that the references provided by the Applicants have been discussed in detail in Paper No. 14, for example, Elliot, et al. teach that the 65 kDa isoform of GAD "is implicated in autoimmune diabetes". (see the abstract) and the treatment of NOD mice with the 67kDa isoform of GAD. The use of the NOD mouse model is discussed below, and the 67 kDa isoform is not claimed in the instant application. The Examiner maintains that the record is clear that she has presented numerous references in support of her position that the instant invention is not supported by an enabling disclosure.

21. The Applicants' comments regarding the Harrison article are not persuasive, since the clinical trial presented by Applicants was only a limited toxicology trial, of unknown duration, and not providing therapy information, as discussed, *supra*.
22. The Applicants also present additional arguments concerning the Petersen reference and alleging that the Examiner did not address Applicants arguments as to why the BB rat was a less useful model for the treatment of IDDM than the NOD mouse. However, in Paper No. 18, the Examiner stated: "In contrast, the Examiner was merely demonstrating that in a more recent (1997) publication, a different animal model teaches that treatment with GAD65 does not protect against diabetes and Petersen teaches that "neither GAD65 nor BSA autoimmunity is important for the development of diabetes in BB rats, in contrast to the situation in NOD mice, and further emphasizes that extrapolation from only one animal to autoimmune diabetes in general may not be appropriate." (see the abstract). Since the Applicants own disclosed experiments used rat islet cells (see Figure 1 of the specification), the Examiner maintains that it is an appropriate model, absent any evidence provided by the Applicants that the BB rats model is not predicative of human disease. The reference also teaches that "One of the limitations in extrapolating data from an animal model like the NOD mouse, is that it represents only one genotype, whereas IDDM in humans in all probability has a much more multifaceted etiology, pathogenesis and genetic predisposition." (see page 130, col. 1). Applicants admit on page 5 of the response in Paper No. 17, that 20% of IDDM patients presumably have a different genetic predisposition." The Examiner addressed the Applicants arguments concerning the two models by providing a teaching by Petersen which sheds doubt on the NOD mouse model. Additionally, Applicants assertions in Paper No. 17, that because the BB rat does not produce antibodies against GAD makes it an unreliable model for human IDDM is not found convincing because other aspects of NOD mouse diabetes are not similar to humans, such as the unequal distribution among males and females and the lack of pancreatitis in NOD mice. Also, in support of the Examiner's position, Atkinson and Leiter (*Nature Med.* 5(6):601-604, 1999) further state that when the NOD mouse model "is used as a surrogate for humans, genus-specific differences that restrict their interpretation are unavoidable." (see page 601, col.2, last paragraph) Also, Atkinson state the "GAD autoimmunity does not seem to be a factor in BB rats, emphasizing the point that many models should be evaluated before extrapolation to humans are attempted." (see page 603, col. 2, last paragraph).
23. It is noted that the instant specification does not disclose the use of NOD mice or any other animal model is disclosed in the instant specification.

24. Claims 60 and 61 stand rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention. There is no support in the specification or claims as originally filed for a method for preventing or delaying the development of clinical symptoms of insulin dependent diabetes, as recited in claim 60. The claim differs in scope with the disclosure in the instant case. Applicants are invited to provide the Examiner with the location in the specification where the above wording is supported. **This is a new matter rejection.**

25. The Applicants argue that claims 60 and 61 are supported and that the terms "preventing or delaying" rather than "inhibiting" and "delaying" are used synonymously in the two claims. The Applicants further state that the recitations "patient" and "animal" is "merely a difference in semantics" and that the recitation of "essentially pure" would be merely a difference in terminology over the "homogenous peptides of at least 99% w/w" disclosed in the specification. However, the Examiner maintains that these new recitations are of a different scope than disclosed in the instant specification and thus constitute new matter. The case law recited by Applicants is not persuasive because the Applicants are not permitted to change the scope of the original disclosure by amendment. *see MPEP 706.03(o)*.

Claim Rejections - 35 U.S.C. § 102

26. The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

A person shall be entitled to a patent unless --

(e) the invention was described in a patent granted on an application for patent by another filed in the United States before the invention thereof by the Applicant for patent, or on an international application by another who has fulfilled the requirements of paragraphs (1), (2), and (4) of section 371(c) of this title before the invention thereof by the Applicant for patent.

27. Claim 31 stands rejected under 35 U.S.C. 102(e) as being anticipated by Atkinson (US Patent No. 5,762,937).

28. The '937 patent teaches a method for inhibiting the development of IDDM comprising the administration of GAD to a patient is taught in col. 4, lines 40-48 and col. 25 line 53 and bridging over to col. 26, line 14. The administration of a therapeutically-effective dosage is inherent in the successful treatment of any disease. Additionally, claims 1 and 2 teach the claimed invention. Therefore, the reference teachings anticipate the claimed invention.

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29. It is noted that the Applicants have not provided any arguments regarding the outstanding rejection under 35 U.S.C. 102, and that the rejection over the '937 patent can be resolved by interference. The Applicants also stated that "if the Examiner is of the view that any of the presently pending claims are patentably distinct over Atkinson, Applicants will provide declarations to overcome the cited reference on the basis of derivation. The Examiner refers the Applicants to the written record of the position of the Examiner.

Claim Rejections - 35 U.S.C. § 103

30. The following is a quotation of 35 U.S.C. § 103 which forms the basis for all obviousness rejections set forth in this Office action:

A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negated by the manner in which the invention was made.

Subject matter developed by another person, which qualifies as prior art only under subsection (f) or (g) of section 102 of this title, shall not preclude patentability under this section where the subject matter and the claimed invention were, at the time the invention was made, owned by the same person or subject to an obligation of assignment to the same person.

31. It is acknowledged that the Applicants have stated in Paper No. 13 that the subject matter of all claims was commonly owned by the University of California and Yale University.

32. Claims 35 and 54-57 and new claims 62 and 63 stand rejected under 35 U.S.C. 103(a) as being obvious over Chang and Gottlieb (*J. Neurosci.* 8(8):2123-2130, 1988).

33. Chang and Gottlieb (*J. Neurosci.* 8(8):2123-2130, 1988) teach the use of GAD in a pharmaceutical preparation by immunizing a mouse with purified rat brain GAD in a composition comprising GAD and Freund's adjuvant (see page 2124, col. 2, paragraph 3, in particular). Chang and Gottlieb do not teach a composition comprising GAD in a pharmaceutically-acceptable carrier for use in humans. However, one of ordinary skill in the art at the time the invention was made would have been motivated to provide a pharmaceutically-acceptable carrier for humans in light of the teaching by Chang and Gottlieb of a pharmaceutically-acceptable carrier for use in rats. Additionally, pharmaceutical carriers are well known to one of ordinary skill in the art for use in humans. Claims 54-56 are included because a product is a product, regardless of its source. From the combined teachings of the references, it is apparent that one of ordinary skill in the art would have had a reasonable expectation of success in producing the claimed invention. Therefore, the invention as a whole is *prima facie* obvious to one of ordinary skill in the art at the time the invention was made, as evidenced by the references, especially in the absence of evidence to the contrary.

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34. The Applicants argue that the mere knowledge of the existence of a pharmaceutically acceptable carrier for humans does not provide motivation for one to use such a carrier in place of the Freund's adjuvant used by Chang & Gottlieb." The Applicants further state that the only reason why one would have been motivated to use a pharmaceutical carrier for use in humans is if one intended to use GAD for administration to humans. However, the Examiner respectfully reminds Applicants that the claims are drawn to a patient, and by Applicants own statements on page 7, paragraph 29 of the response, animals can be patients as well as humans. In light of the teaching of a pharmaceutical composition for use in rats, recognized as a animal by one of ordinary skill in the art, the pharmaceutical composition would have been obvious to one of ordinary skill in the art at the time the invention was made.

Conclusion

35. The prior art made of record and not relied upon is considered pertinent to Applicant's disclosure. US Patent No. 5,998,366 (Tobin, et al.) claims a method for ameliorating a GAD associated autoimmune disorder comprising the administration to the patient of a GAD65 peptide consisting of a series of specific recited peptides.

36. Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 C.F.R. 1.136(a).

37. A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 C.F.R. 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.


38. Papers related to this application may be submitted to Group 1640 by facsimile transmission. Papers should be faxed to Group 1640 via the PTO Fax Center located in Crystal Mall 1. The faxing of such papers must conform with the notice published in the Official Gazette, 1096 OG 30 (November 15, 1989). THE CM1 FAX CENTER TELEPHONE NUMBER IS (703) 305-3014 or (703) 308-4242.

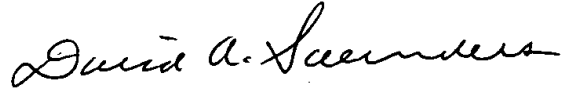
39. Any inquiry concerning this communication or earlier communications from the Examiner should be directed to Mary Tung whose telephone number is (703)308-9344. The Examiner can normally be reached Tuesday through Friday from 8:30 am to 5:30 pm, and on alternating Mondays. A message may be left on the Examiner's voice mail

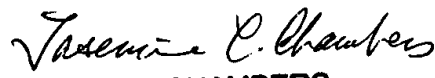
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service. If attempts to reach the Examiner by telephone are unsuccessful, the Examiner's supervisor, Christina Chan can be reached on (703) 308-3973. Any inquiry of a general nature or relating to the status of this application should be directed to the Group 1640 receptionist whose telephone number is (703) 308-0196.


April 20, 2001
Mary B. Tung, Ph.D.
Patent Examiner
Group 1640


DAVID SAUNDERS
PRIMARY EXAMINER
ART UNIT ~~182~~ / 644


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